

59 ± 11 years (69% males). History of previous MI was present in 45% of the pts and 13% presented with cardiogenic shock. The culprit lesion was located in the LAD (23%), RCA (37%), LCX (11%) or a saphenous vein graft (28%). Adjunctive percutaneous revascularization was performed in 93% of the patients: PTCA alone (46%), coronary stenting (46%), and rotational atherectomy (1%). Percent diameter stenosis was reduced from 82% to 50% after Angiojet, and to 24% after definitive treatment ($P = 0.0001$).

Procedural success, defined as the restoration of TIMI 3 flow and in-hospital freedom from death, emergent bypass surgery or major disabling stroke, was accomplished in 98%. Procedure complications included distal embolization (2%), transient no-reflow (2%), and abrupt closure (2%). At 1 month clinical follow up, 2 (2%) pts died; 3 (3%) patients had recurrent MI and 2 (2%) required repeated target vessel percutaneous revascularization. At 1 month follow up 82 (92%) patients were alive and free from recurrent MI and repeat revascularization procedure.

Conclusions: The Angiojet is a promising adjunctive device for establishing patency in thrombus laden lesions. It is of particular utility in acute myocardial infarction where the presence of coronary thrombi enhances the periprocedure complication rate.

1161-151 Liposomal PGE-1 Adjunctive Treatment for Acute Myocardial Infarction: Final Results of the LIFT Pilot Trial

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Background: Liposomal PGE-1 (TLC-C53) inhibited platelets and ICAM-1 expression which accelerated thrombolysis and reduced infarct size in animal myocardial infarction models.

Methods: To evaluate the effect of TLC-C53, an investigational drug, on thrombolysis and infarct salvage. 121 patients with myocardial infarction (MI) were randomized to receive either TLC-C53 (bolus plus 5 additional doses/48 hours) or placebo just prior to front-loaded t-PA. Coronary angiography was performed at 30, 60 and 90 minutes and 5-7 days after admission. Left ventriculography was performed acutely and 5-7 days post MI. Angiograms were analyzed for TIMI flow, global and regional left ventricular function.

Results: Patients were well matched for age, weight and time to treatment. There was a trend toward better early TIMI 3 patency in the TLC-C53 patients at 60 minutes post treatment (53 vs 47%, $p = NS$). However, at 90 minutes the TIMI 3 patency was equivalent (54 vs 55%). There was also a trend toward lower reinfarction and re-occlusion in the TLC-C53 group compared to placebo (2 vs 11% respectively; $p = 0.06$). There were no differences in acute or follow-up ejection fraction or regional function between the groups.

Conclusion: TLC-C53 did not produce dramatic improvements in infarct salvage or vessel patency at the dosage level and frequency evaluated however it may reduce recurrent ischemia and reinfarction.

1161-152 Phase I Safety Trial of Soluble Complement Receptor Type 1 (TP 10) in Acute Myocardial Infarction

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Background: Complement activation occurs in acute MI and may contribute to reperfusion injury. Thus complement inhibition might reduce injury following PTCA or thrombolysis for MI. This phase I trial tested the pharmacokinetics (PK) and safety of the complement inhibitor TP10 (T Cell Sciences, Needham, MA), a recombinant soluble form of human erythrocyte complement receptor 1.

Methods: TP10 was given by open label, ascending dose schedule (0.1, 0.3, 1, 3, or 10 mg/kg i.v. over 30 min.) to 24 patients receiving lytics ($n = 9$) or primary PTCA ($n = 15$) for first MI. CH50, C3a, and C4a were measured at 0, 8, 24, 48, and 168 hr.

Results: There were no immunogenic responses or serious adverse effects directly attributed to TP10. Non-compartmental PK analysis showed TP10 $t_{1/2} = 94.5 \pm 57.5$ hr. TP10 caused dose ($p < 0.05$ for 0.1 vs higher doses) and time-dependent ($p < 0.05$ for 8 and 24 hr vs 0 and 168 hr) decrease of CH50 (2 way ANOVA). C3a tended to fall at 8 h relative to 0 h ($p = ns$) at all TP10 doses, in contrast to the predicted rise with MI, suggesting complement inhibition. As expected based on the mechanism of action of TP10, there was no time or dose effect on C4a. In patients receiving lytics there was a trend toward lower CKMB (normalized to ECG area at risk) in those receiving higher (> 1.0 mg/kg) vs lower dose TP10 (52 vs 511, $p = 0.06$).

Conclusion: TP10 is well tolerated and non-immunogenic in MI. The expected rise in C3a during MI was not seen at any dose of TP10, suggesting effective complement suppression in these patients.

1161-153 A K⁺-ATP Channel Opener, Nicorandil, Limited Infarct Size in Patients With Acute Myocardial Infarction

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Background: We have recently reported that nicorandil (NCR), a K⁺-ATP channel opener, restored blood flow to the reperfused myocardium in patients (pts) with acute myocardial infarction (AMI). However, it is unknown about the infarct size-limiting effect of NCR. Thus we sought to investigate whether NCR limit infarct size in pts with their first anterior AMI.

Methods: After successful coronary reflow, 15 pts received intracoronary administration of NCR (2 mg), and 18 pts received placebo. Peak serum creatine kinase (CK) levels, defect volume index (DVI) determined by thallium-201 SPECT, global ejection fraction (EF) and left ventricular end-diastolic volume index (EDVI) in the acute stage and one month after the onset were compared between the two groups of patients.

Results: 1) There were no differences between the two groups in age, gender and time to reperfusion (4.2 ± 2.2 vs 4.6 ± 1.8 hrs, $p = 0.64$). 2) Peak CK (2248 ± 1386 vs 3642 ± 1624 U/L, $p = 0.03$) and peak CK-MB (191 ± 118 vs 303 ± 122 U/L, $p = 0.03$) levels were significantly lower in NCR group. 3) DVI was significantly lower in NCR group (961 ± 735 vs 1557 ± 781 U, $p = 0.05$). 4) EF was significantly improved in a month in NCR group (42 ± 14 to $52 \pm 16\%$, $p = 0.03$), while it was not in placebo group (44 ± 11 to $47 \pm 13\%$, $p = 0.26$). 5) EDVI did not differ both at the acute stage and a month after the onset.

Conclusion: Intracoronary administration of NCR limited infarct size and improved functional recovery in a month after the onset. Thus intracoronary nicorandil administration seems to be a useful adjunctive therapy following successful coronary reperfusion in patients with AMI.

1162 Diagnostic Methods of Detecting Ischemia

Wednesday, April 1, 1998, 9:00 a.m. - 11:00 a.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 10:00 a.m. - 11:00 a.m.

1162-131 Beneficial Effects of Troglitazone in Vasospastic Angina Pectoris With Diabetes Mellitus

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Backgrounds: The relationships between insulin resistance and coronary artery disease have a great deal of attention.

Methods: In order to investigate whether troglitazone, recently used for insulin resistance, has impacts upon clinical manifestations of coronary artery disease we administered troglitazone for four months in diabetic patients with angiographically documented coronary vasospasm and residual angina pectoris even with conventional medications for vasospastic angina pectoris ($n = 8$). At baseline and at four months after medication, we assessed anginal episodes and non-invasively measured the reactive changes in lumen diameter of right brachial artery following transient occlusion for five minutes (FMD; flow-mediated endothelium-dependent vasodilation), and after sublingual administration of (300 µg) glyceryl trinitrate (GTN; endothelium-independent vasodilation).

Results: Duration of anginal episodes (AP; minutes per month) were significantly reduced after medication. FMD was improved after medication. GTN was similar before and after medication. Reduction of AP was correlated to improvement of FMD ($r = 0.6$, $p < 0.01$).

	premedication	postmedication	p value
AP	72 ± 68	14 ± 17	0.04
FMD	4.3 ± 1.4%	6.6 ± 1.6%	0.03
GTN	11.5 ± 4.3%	13.3 ± 2.9%	n.s.

Conclusions: These findings suggest that troglitazone improves endothelium-dependent vasoreactivities and reduces anginal episodes in diabetic patients with vasospastic angina pectoris, and insulin-resistance may contribute to clinical manifestation of vasospastic angina pectoris with diabetes mellitus.